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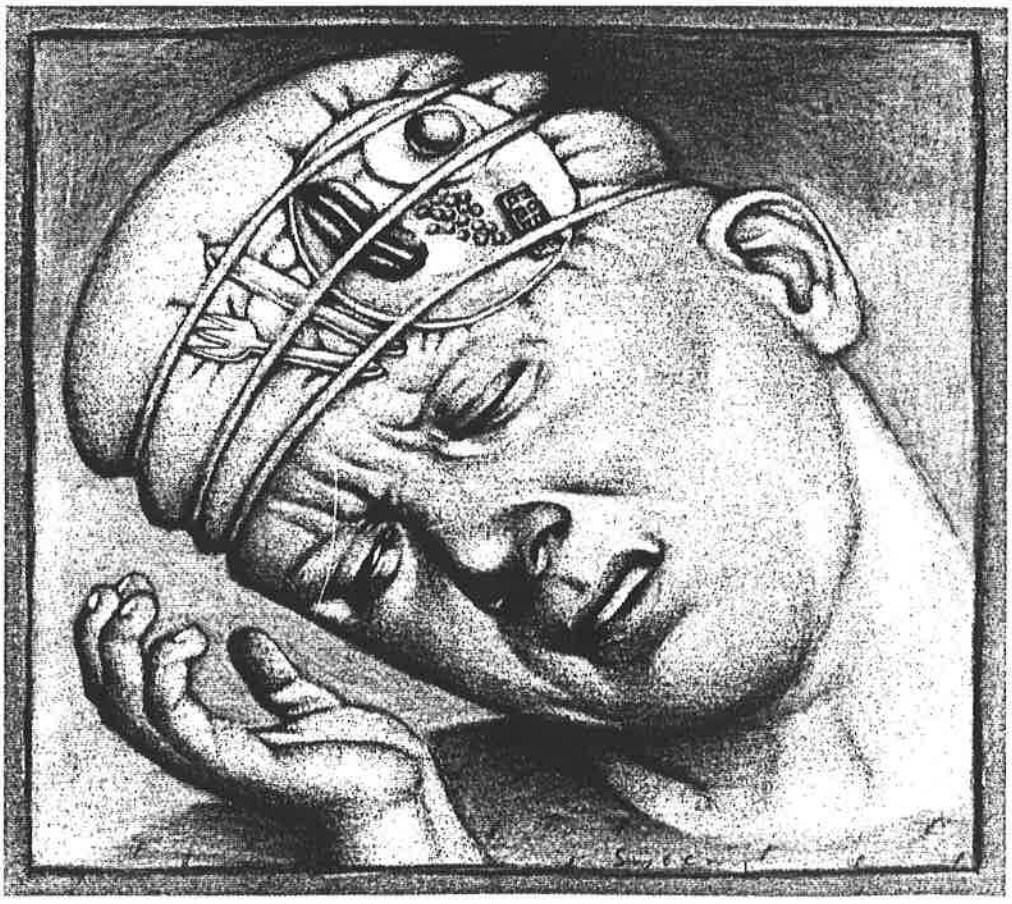

A PGM symposium on urinary incontinence
Second of four articles

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Female urinary incontinence

Therapeutic approaches

Preview questions

What are the causes of bladder- and sphincter-related incontinence?

When should augmentation cystoplasty be used to treat bladder-related incontinence?

What is the goal when treating intrinsic sphincter incompetence?

Urinary incontinence has been and continues to be a problem of epic proportion, affecting more than 20 million Americans, prompting more than 50% of nursing home admissions, and fostering a $1 billion pad and appliance industry.[1] Defined as the involuntary loss of urine from the urethra, urinary incontinence can be caused by dysfunction of the bladder or the sphincter (table 1). (Urinary leakage, or loss of urine from a site other than the urethra, refers specifically to fistula and ureteral ectopia and is beyond the scope of this article.)

Bladder-related incontinence

Bladder-related incontinence may be due to reduced capacity, instability, poor compliance, or incomplete emptying. A bladder of reduced "organic" capacity (contracted bladder) can be caused by tuberculosis, chemical cystitis, radiocystitis, end-stage interstitial cystitis, and neurogenic impairment. A bladder of satisfactory organic capacity but reduced "functional" capacity can result from neurogenic impairment and idiopathic detrusor instability (unstable bladder). McGuire (personal communication) has aptly characterized the unstable bladder as "a bladder that contracts without the permission of its owner." Regardless of the specific cause, a reduction in the organic or functional capacity of the bladder causes a dysfunction of urine storage that results in urgency, frequency, and incontinence.

In general, a bladder of significantly reduced organic capacity does not respond to conservative approaches, such as anticholinergic therapy, behavior modification, and bladder retraining techniques. In contrast, a bladder of reduced functional capacity is more likely to benefit from cholinolytic therapy and behavior modification, including fluid restriction, timed voiding with postponement of micturition, and exercises to strengthen pelvic-floor muscles.

continued

If pharmacologic therapy and behavior modification fail in the treatment of bladder-related incontinence, other approaches may be tried, including augmentation cystoplasty.

Table 1. Causes of the two major types of female urinary incontinence

Bladder-related
Reduced capacity
Instability
Poor compliance
Incomplete emptying
Sphincter-related
Anatomic malposition of intact sphincter unit
Intrinsic sphincter damage
Multiple failed surgical procedures for incontinence
Pelvic trauma, including surgery, irradiation, and fracture
Sacral arc lesions
Neuropathic urethral dysfunction

Table 2. Management of bladder-related incontinence after pharmacotherapy and behavior modification fail

Sacral root stimulation
Rhizotomy
Phenol injection
Bladder denervation procedures
Augmentation cystoplasty

If pharmacologic therapy and behavior modification fail, alternative approaches to treatment (table 2) include the following.

- Sacral root stimulation—This technique involves either percutaneous or open surgical implantation of electrodes around the second and third sacral roots to manipulate detrusor and pelvic-floor contractility in such a way as to favorably affect incontinence. It is being used by Tanagho (personal communication), who has reported the rate of cure or improvement to be in the 50% to 75% range. Because it increases urethral resistance, sacral root stimulation is also being used to treat sphincter-related incontinence. At this juncture, however, this approach remains largely a laboratory technique.
- Rhizotomy—This neurosurgical ablative procedure involves the selective cutting of sacral roots subserving detrusor function.
- Phenol injection—Endoscopic subtrigonal phenol injection chemically ablates the neural plexus at the base of the bladder.
- Bladder denervation procedures—Most of the numerous bladder denervation procedures attempted thus far have produced equivocal results at best, probably because of the impossibility of completely denervating the bladder and the phenomenon of reinnervation with sprouting of nerve fibers.
- Augmentation cystoplasty—With this technique, bladder capacity is enlarged by anastomosing a segment of bowel to the bladder.

Until neurophysiologic and technical problems with sacral root stimulation are resolved and this technique becomes time-tested and is in wide clinical use, we prefer augmentation cystoplasty for treating bladders of reduced functional or organic capacity.

The segment of bowel selected for augmentation will not affect the ultimate results as long as certain principles are strictly adhered to.[2] The bowel segment must be detubularized to favorably modify its contractility. Rather than the loop being left intact, the circular fibers of the bowel segment must be transected to improve compliance. The bowel segment must be of sufficient size to allow for the creation of a capacious reservoir and must be carefully mobilized so that it has an adequate vascular supply and no tension is placed on the mesentery. The bowel-bladder anastomosis should be wide and stricture-free. To avoid stress urinary incontinence in patients with a poorly supported anterior vaginal wall, bladder neck suspension must be performed before completion of the operation.

Although generally proved effective, augmentation cystoplasty is a major procedure not without complication or risk.

Bladder instability is triggered by tension receptors in the bladder wall that facilitate the normal brainstem micturition reflex. Augmentation cystoplasty seems to delay initiation of the facilitated reflex, allowing the patient to defer micturition and hold urine for three to four hours without urgency or incontinence.

In our experience at UCLA, augmentation cystoplasty cures bladder-related incontinence in 75% to 85% of patients. However, 10% to 20% may require self-catheterization, which is a better alternative than incontinence and the wearing of perineal pads.

Although generally proved effective, augmentation cystoplasty is a major procedure not without complication or risk. Chronic renal insufficiency is a relative contraindication to augmentation because of the risk of absorption of urinary substrates from the absorptive surface of the cystoplastic segment, with the potential for metabolic and electrolyte abnormalities. Also, any complication common to bowel surgery can occur, including ileus, bowel obstruction, anastomotic leak, hernia, and pelvic abscess. Thus, the surgeon must be committed to long-term follow-up.

Sphincter-related incontinence

Sphincter-related incontinence may be caused either by anatomic malposition of an intact sphincter unit or by intrinsic sphincter damage when, with or without an accompanying anatomic abnormality, the bladder neck no longer functions as an intact sphincter unit.

ANATOMIC INCONTINENCE—In a normal woman in the supine position, urethral pressures range from 40 to 80 cm H_2O and intraabdominal pressures range from 5 to 10 cm H_2O. Although coughing or stress maneuvers cause an increase of 20 to 100 cm H_2O or more in abdominal and bladder pressures, urinary incontinence does not occur in the normal female for several reasons: (1) A reflex contraction of the pelvic floor muscles serves to increase urethral resistance. (2) Because the urethra is in a well-supported retropubic position, the increased pressure is effectively transmitted to both the bladder and the urethra, thereby maintaining the pressure gradients. (3) The locus of force generated is experienced not at the bladder neck, which is in a nondependent position, but at the bladder base, which is the most dependent structure.

With prolapse and hypermobility of the bladder neck, the compensatory and protective mechanisms just described fail. Because the bladder neck drops out of the realm of effective pressure transmission and assumes the most dependent position, the locus of force generated is aimed directly at the bladder neck, and stress incontinence ensues.

CLINICAL PRESENTATION—Childbirth, hysterectomy, menopause, and weakened pelvic support contribute to the genesis of prolapse and hypermobility and, in turn, stress incontinence. Thus, the patient who typically needs bladder neck suspension is a multiparous, middle-aged, postmenopausal woman with progressive symptoms of urine loss on stress maneuvers. Examination of the vaginal wall often reveals urethral hypermobility concomitant with cystocele, rectocele, and perineal floor laxity, since pelvic-floor disease is not restricted to the anterior vaginal wall but is a tubular phenomenon. A spurt of urine from the urethra is typically observed on Valsalva's maneuver or coughing.

DIAGNOSTIC TESTS—Urodynamically, genuine stress incontinence is defined as the loss of urine from the urethra during an elevation in intraabdominal pressure without a true detrusor contraction. This is easily demonstrated by cystometry, in which rectal and intravesical pressures are monitored. Urodynamic studies are useful in assessing functional bladder capacity, determining postvoiding residual urine volumes, and documenting the

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Cystourethroscopy using a 0° lens and a urethroscope is a fundamental component of the diagnostic workup for sphincter-related incontinence due to an anatomic abnormality.

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presence or absence of detrusor instability. Uroflowmetry often demonstrates "superflows." We have not found urethral closure-pressure profilometry to be of any clinical value.

Cystourethroscopy using a 0° lens and a urethroscope is a fundamental component of the diagnostic workup. Because of the presence of urethral descensus, the urologist often has to arc the urethroscope in a downward direction to facilitate its passage. The "Q-tip" test (placing a cotton-tipped applicator in the urethra and measuring the angle off the horizontal) similarly demonstrates the angulation created by the urethrocele. When the urethroscope is positioned at the level of the bladder neck and proximal urethra, Valsalva's maneuver demonstrates hypermobility. Bladder-base hypermobility can be assessed in a similar manner. In addition, urethroscopy reveals the degree of urethral vascularity and coaptation, and examination with a 70° lens rules out concurrent bladder disease. In any case of incontinence, ureteral ectopia and vesicovaginal fistula must be ruled out.

Voiding cystourethrography with anteroposterior, oblique, and lateral films in both relaxing and straining modes is an important diagnostic adjuvant. We consider this study to be the equivalent of physical examination of the bladder with the patient in the standing position. It demonstrates the degree of urethral and bladder-base hypermobility, funneling of the bladder neck, and the degree of cystocele. Also, it objectively shows stress incontinence and the amount of postvoiding residual urine present.

SURGICAL TREATMENT—When genuine stress incontinence is associated with urethral hypermobility, the goal of surgery is to elevate and reposition

The most popular procedure for genuine stress incontinence associated with urethral hypermobility is Kelly plication.

the bladder neck and proximal urethra into a high, well-supported retropubic position. Surgical alternatives include an abdominal or a vaginal approach.

The most popular procedure performed to correct this problem is Kelly plication, which was described in the early 20th century[3] and remains widely in vogue today, predominantly among gynecologists. Kelly plication involves a vaginal incision and placement of several sutures in periurethral tissue to buttress and reef the bladder neck. While this procedure corrects prolapse, its success rate for curing incontinence is only 50% because of failure of the plication to restore the bladder neck to a well-supported retropubic position.

The abdominal approach is exemplified by the Marshall-Marchetti-Krantz[4] and Burch[5] procedures. The Marshall-Marchetti-Krantz procedure was the first of the retropubic "pin-up" operations and has yielded satisfactory results with respect to curing incontinence. In this procedure, several pairs of chromic suture are used to pin the upper wall of the vagina and the lateral wall of the urethra to the pubic periosteum. In the Burch procedure, which is similar in principle to the Marshall-Marchetti-Krantz procedure, perivaginal fascia is pinned to Cooper's ligament.

Almost 30 years ago Pereyra[6] described the original needle suspension procedure, an alternative to the abdominal suspension procedures. Since then, numerous modifications and variations have been reported in the urologic literature. However, the principle remains the same: Through a transvaginal approach, the bladder neck is suspended by suspension sutures that anchor tissue adjacent to the bladder neck to the abdominal fascia; the sutures, which are placed transvaginally, are transferred to the abdomen by means of a special needle (ligature carrier), thus avoiding the need to open the abdomen.

Given the multitude of different procedures available, what is a reasonable choice? Is a vaginal approach superior to an abdominal approach? The answers to these questions can be obtained by comparative analysis of the results and the advantages and disadvantages of the individual techniques. The various techniques are remarkably comparable in resolving stress incontinence. Clearly, if similar results can be achieved with abdominal and vaginal approaches, it would be in the best interest of the patient to choose the procedure that results in the least pain, morbidity, and risk and in the shortest hospital stay and the most rapid return to a productive life-style. In this regard, vaginal needle suspension procedures confer a distinct advantage. Also, the ability to simultaneously correct concomitant vaginal disease favors the vaginal approach. The choice of a particular vaginal technique is of little importance as long as the following basic principles are strictly adhered to:

- Mobilization of urethra and bladder neck from fixation and tethering so that suspension without tension can be achieved (especially important in secondary procedures)
- Use of strong anchoring tissue
- Precise placement of suspension sutures (placement that is too distal results in urethral kinking, too proximal in ineffective suspension, too medial in urethral obstruction, and too lateral in ineffective suspension and possibly pain from a suture penetrating the levator ani group of muscles)
- Adequate securing of anchoring tissue (with helical sutures seeming to have advantages over buttress or loop)
- Fingertip guidance of passage of ligature carrier
- Use of double-pronged ligature carrier (to reduce the number of passes and provide a fascial "pledget")
- Cystoscopic control (to confirm that the bladder and urethra have not been penetrated and to en-

continued

An enterocele may be a sequela of repositioning the bladder neck, since the cul-de-sac after suspension is in a position disposing to small-bowel herniation.

Table 3. Surgical management of sphincter-related incontinence

Anatomic incontinence
Kelly plication
Abdominal bladder neck suspension
Vaginal bladder neck suspension
Sacral root stimulation

Intrinsic damage
Periurethral Teflon injection
Implantation of artificial urinary sphincter
Sling procedures

sure that the bladder neck is properly suspended and ureteral efflux is present)

- Tying of sutures over fascia with proper amount of tension

Any procedure that can satisfy the aforementioned criteria works, providing the proper diagnosis has been made. Results are satisfactory in about 90% of patients.[7] However, patients must be clearly apprised of the potential for persistent urgency, despite the cure of stress incontinence, and forewarned of the possible necessity for extended bladder drainage before spontaneous voiding resumes. An enterocele may be a sequela of repositioning the bladder neck, since the cul-de-sac after suspension surgery may be in a position disposing to small-bowel herniation. Also, the potential exists for fistula and for injury to the ureter, bladder, or urethra. In a small number of patients, the procedure fails.

Recovery from needle vesicourethral suspension procedures is typically rapid and uneventful. After the Raz modification,[8] oral intake of fluids and food is permitted as soon as the patient can tolerate it. Antibiotics are administered parenterally for 24 hours postoperatively and orally for several days thereafter. On the first postoperative day, the intravenous line, urethral Foley catheter, and vaginal packing are removed, and ambulation is encouraged. About 50% of patients void, with residual urine amounting to less than 75 ml, on the first postoperative day; 90% void by the end of the first week.[9] If the interval between surgery and the resumption of spontaneous voiding is prolonged, use of a suprapubic tube (a Foley catheter placed into the bladder through the anterior abdominal wall at the time of surgery) is a simple means of outpatient management. The suprapubic tube is removed when spontaneous voiding resumes and residual urine volumes become negligible. After uncomplicated surgery for anatomic incontinence, patients generally are discharged from the hospital on the second postoperative day.

INTRINSIC DAMAGE—The predominant cause of intrinsic sphincter incompetence is failed surgery for incontinence, especially when multiple anterior vaginal procedures result in scarring of a fixed, open urethra in a normal position. Pelvic trauma due to surgery, irradiation, or fracture is another cause, as are sacral arc lesions or neuropathic urethral dysfunction, which results in bladder neck incompetence. Intrinsic sphincter incompetence occurs as a result of direct damage to the urethral musculature or periurethral tissue, urethral ischemia, or damage to the pelvic nerves subserving urethral function.

CLINICAL PRESENTATION—The patient with intrinsic sphincter damage presents with severe stress incontinence with leakage of urine in the upright and supine positions. Essentially, leakage occurs regardless of activity or position. Urethroscopy demonstrates an open bladder neck in the absence of a detrusor contraction. Hypermobility of the proximal urethra and bladder neck may also be present but usually is not. The endoscopic appearance of the urethra gives rise to the descriptive terms "lead-pipe" and "pipestem," denoting rigidi-

Treatment of intrinsic sphincter incompetence entails increasing urethral resistance by providing urethral compression.

ty and scarring. Urethral closure-pressure profilometry demonstrates low pressures.

THERAPEUTIC ALTERNATIVES—Treatment of intrinsic sphincter incompetence entails increasing urethral resistance by providing urethral compression. This can be accomplished by periurethral Teflon injection,[10] implantation of an artificial urinary sphincter,[11] or a sling procedure (table 3).

Teflon, a compound of pastelike consistency, is injected under endoscopic visual control into the submucosal urethral wall in circumferential fashion to create coaptation of the urethra. This simple technique can be done in the office using only a local anesthetic and may be repeated without jeopardizing the chances for success of other surgical alternatives. Teflon injection has a 50% to 65% success rate but has been associated experimentally with migration of particles.[12]

Another alternative in selected cases is implantation of an artificial urinary sphincter through an abdominal approach. This semiautomatic mechanical device consists of an inflatable cuff placed around the bladder neck, a reservoir implanted in the preperitoneal space of the abdomen, and a pump implanted in the subcutaneous tissue of the labia majora. Fluid within the cuff that surrounds the bladder neck keeps the patient continent. When the patient desires to urinate, she compresses the pump, which results in transfer of fluid from the cuff to the reservoir and hence in deflation of the cuff. We have not used a sphincter to treat female incontinence, as we prefer a vaginal sling procedure.

Sling procedures have the advantages of comparative simplicity, documented efficacy, and no requirement for foreign prosthetic material. However, the plethora of sling procedures described in the medical literature attests to the lack of one ideal method, which should be not only simple but reliable and reproducible in the hands of any urologist,

continued

The harvesting of autogenous tissue for use as a sling via an incision that has to be made anyway is better than any harvesting technique that involves a secondary incision.

incur minimal morbidity, and necessitate only a short hospital stay. The many reported procedures differ from one another only in the choice of material selected for the sling and the tissue to which the sling is anchored.

Certainly, autogenous tissue offers a distinct theoretical advantage over any synthetic material because of the lessened risk of infection or inflammation associated with the presence of a foreign body. The harvesting of autogenous tissue for use as a sling via an incision that has to be made anyway (ie, rectal fascia obtained via a retropubic incision for a retropubic pubovaginal sling) is better than any harvesting technique that involves a secondary incision (ie, fascia lata or palmaris longus).

Our sling technique,[13] which utilizes an anterior vaginal wall island lying directly underneath the urethra, is unique in that it requires no extravaginal harvesting incision and retains the vascular supply to the sling. Pressure is evenly distributed because the island is in situ, is actually attached to the urethra, and covers a greater amount of urethral surface area. In addition, our procedure involves no dissection directly under the bladder neck and the urethra but rather only lateral dissection, which minimizes the risk of inadvertent urethral or bladder injury. Dissection in the plane between the anterior vaginal wall and the urethra is obviated, as is the mechanical problem of bulky tissue interposed between the urethra and the vagina. This is the only technique described that does not require an extensive abdominal incision, thus minimizing morbidity, causing much less postoperative pain, and hastening recovery and discharge from the hospital.

Any sling technique that works by urethral compression involves the risks of delayed voiding, obstruction, irritative voiding symptoms, de novo instability, and wound or urinary tract infection. The onset of de novo instability, ie, detrusor instability present postoperatively but not preoperatively, is probably related to the relative degree of obstruction created by the sling.

In a series of 32 patients on whom we performed the vaginal sling procedure,[13] all six with neurogenic urethral incompetence had excellent results; all required self-catheterization. Of the 26 patients with nonneurogenic urethral incompetence, 22 (85%) had very good or excellent results. De novo instability was identified in 6 of 26 (23%) patients, who presented with mild irritative symptoms and generally responded well to anticholinergic medication. One (4%) of the 26 patients required catheterization for a nine-month period before spontaneous voiding resumed.

Summary

Urinary incontinence results from bladder or sphincter dysfunction. Bladder-related incontinence due to a reduced-capacity, unstable, or noncompliant bladder can often be remedied by augmentation techniques. For anatomic incontinence, excellent results are achieved with use of any of the retropubic or transvaginal suspension techniques that restore the bladder neck and proximal urethra to a high, fixed retropubic position. However, when the urethral sphincter is intrinsically damaged, mere restoration of position will fail to cure the problem. Such damage warrants use of a compression procedure that increases urethral resistance, such as one of the many sling techniques, implantation of an artificial urinary sphincter, or periurethral Teflon injection. PGM

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Self-assessment test

Select the best answer for each of the following. Answers are given on page 151.

1. Bladder-related incontinence may be due to
   1. Small capacity
   2. Instability
   3. Poor compliance
   4. Incomplete emptying

a. 1, 2, 3
b. 1, 3
c. 2, 4
d. 4
e. 1, 2, 3, 4

2. A bladder of significantly reduced organic capacity responds well to conservative approaches.

a. True
b. False

3. Which of the following statements is/are true of augmentation cystoplasty.
   1. It should be used only as a last resort when conservative treatment fails
   2. It allows the patient to hold urine for three to four hours without urgency or incontinence
   3. Chronic renal insufficiency is a relative contraindication
   4. The segment of bowel selected for augmentation affects the result

a. 1, 2, 3
b. 1, 3
c. 2, 4
d. 4
e. 1, 2, 3, 4

4. Stress incontinence results when the locus of force generated by coughing or stress maneuvers is aimed at

a. The bladder base
b. The bladder neck
c. Either **a** or **b**
d. Neither **a** nor **b**

5. The most popular procedure to correct genuine stress incontinence associated with urethral hypermobility is

a. Kelly plication
b. The Marshall-Marchetti-Krantz procedure
c. The Burch procedure
d. None of these

6. Which of the following statements is/are true regarding vaginal needle suspension procedures?
   1. Recovery is typically rapid and uneventful
   2. Results are satisfactory in about 90% of patients
   3. After uncomplicated surgery, patients are generally discharged from the hospital on the second postoperative day
   4. Extended bladder drainage may be necessary before spontaneous voiding results

a. 1, 2, 3
b. 1, 3
c. 2, 4
d. 4
e. 1, 2, 3, 4

7. Raz's sling technique for treating intrinsic sphincter incompetence is unique in that

a. It requires no extravaginal harvesting incision
b. It retains the vascular supply to the sling
c. It involves no dissection directly under the bladder neck and urethra
d. All of these
e. Only **a** and **b**